

Fat, Hyperlipidemia and Hypercholesterolemia in Diabetic Peripheral Neuropathy

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Abstract

This letter to editor was aimed at expanding the interprofessional understanding on the role of nutrition-induced hematological adaptations such as hyperlipidemia and hypercholesterolemia secondary to fat intake in diet and nutrition in diabetic peripheral neuropathy (DPN). Hypercholesterolemia induced genetic variation in tumor necrosis factor (TNF) receptor 2 gene, High-fat diet fed streptozotocin-induced diabetic rats not only developed peripheral neuropathy faster than their controls [2,3] but also associated disorders like cholesterol-induced gall bladder stones [4] and many intervention studies had shown efficacy for controlling hyperlipidemia and co-existing mechanical and thermal allodynia in experimental models of DPN. Hyperlipidemia is thus to be regarded as a new therapeutic target for DPN.

Keywords: Food; Nutrition; Diet; Hyperlipidemia; Hypercholesterolemia; Diabetic neuropathy.

Dear Sir,

This letter to editor wishes the International Journal of Nutrition and Food Sciences on its maiden journey to scientific excellence to bridge the knowledge-practice gap in the era of scientific evidence-informed practice, education, research and administration both in health and in disease. The authors wish to highlight an example of interprofessional understanding on the role of nutrition-induced hematological adaptations such as hyperlipidemia and hypercholesterolemia secondary to fat intake in diet and nutrition in diabetic peripheral neuropathy (DPN).

Genetic influence was demonstrated by findings of Benjafield *et al* [1] who studied 357

well-characterized white patients and 183 healthy control subjects, and demonstrated genetic variation in tumor necrosis factor (TNF) receptor 2 gene (TNFRSF1B) and its association with hypercholesterolemia as shown by CA16 allele levels tracking with elevation plasma HDL cholesterol.

High-fat diet fed streptozotocin-induced diabetic rats not only developed peripheral neuropathy faster than their controls [2,3] but also associated disorders like cholesterol-induced gall bladder stones [4] and many intervention studies on menhaden oil [5] and *Emblica officinalis* Gaertn (Amla) [6] had shown efficacy for controlling hyperlipidemia and co-existing mechanical and thermal allodynia in experimental models of DPN.

Vincent *et al* [7] recommended hyperlipidemia to be a new therapeutic target for DPN since it was shown by recent data that established dyslipidemia as a significant contributor to the development of diabetic neuropathy. The author explained oxidative stress mechanisms for metabolic imbalances, which mutually included hyperglycemia and hyperlipidemia, in dorsal root ganglia (DRG) of sensory neurons.

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